WEST Search History

DATE: Thursday, June 06, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L8	L1 and L4 and L5	97	L8
L7	La and L4 and L5	4370	L7
L6	L1 and L2 and L3	3	L6
L5	oligonucleotide	42687	L5
L4	antisense	21119	L4
L3	vascular injury	1381	L3
L2	ischemic tissue	909	L2
L1	egr-1	159	L1

END OF SEARCH HISTORY

FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 15:39:33 ON 12 JUN 2002

L1	13954	VASCULAR INJURY
L2	4040	ISCHEMIC TISSUE
L3	4523	EGR-1
L4	129	EARLY GROWTH RESPONSE FACTOR-1
L5	0	L1 AND L2 AND L3 AND L4
L6	4	L1 AND L2 AND L3
L7	0	L1 AND L2 AND L4
L8	107829	REPERFUSION
L9	4	L1 AND L2 AND L3 AND L8
L10	49	L1 AND L3
L11	8	L2 AND L3
L12	71657	ANTISENSE
L13	199	L3 AND L12
L14		

=> d fhitstr ibib abs 1-4 kwic

L3 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2002 ACS
IT **196222-26-1**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)
RN 196222-26-1 CAPLUS
CN DNA, d(C-T-T-G-G-C-C-G-C-T-G-C-C-A-T) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
ACCESSION NUMBER: 2001:319765 CAPLUS
DOCUMENT NUMBER: 134:344560
TITLE: Sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy
INVENTOR(S): Khachigian, Levon Michael
PATENT ASSIGNEE(S): Unisearch Ltd., Australia
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030394	A1	20010503	WO 2000-AU1315	20001026

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: AU 1999-3676 A 19991026
AB The present invention relates to a method for the treatment of tumors, the method comprising inhibiting angiogenesis in a subject in need thereof characterized in that angiogenesis is inhibited by administering to the subject an agent which inhibits induction of EGR, an agent which decreases expression of EGR or an agent which decreases the nuclear accumulation or activity of EGR. The present invention also relates to a method of screening for agents which inhibits angiogenesis. In particular, the invention provides sequences of **antisense oligonucleotides** and catalytic DNA targeting EGR-1 mRNA and their uses in cancer therapy.
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy
AB The present invention relates to a method for the treatment of tumors, the method comprising inhibiting angiogenesis in a subject in need thereof characterized in that angiogenesis is inhibited by administering to the subject an agent which inhibits induction of EGR, an agent which decreases expression of EGR or an agent which decreases the nuclear accumulation or activity of EGR. The present invention also relates to a method of

screening for agents which inhibits angiogenesis. In particular, the invention provides sequences of **antisense oligonucleotides** and catalytic DNA targeting EGR-1 mRNA and their uses in cancer therapy.

ST DNAzyme **antisense oligonucleotide** sequence Egr1 mRNA inhibitor anticancer

IT Quaternary structure
(DNA **triplex**, for decressing Egr-1 gene expression; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT mRNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(EGR-1; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Egr-1, altering expression of; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Egr-1; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT **Ribozymes**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(deoxy; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(double-stranded, of Egr gene; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT **Antisense** RNA
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(for Egr-1 mRNA; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Drug screening
(for angiogenesis inhibitor; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Angiogenesis
Cell migration
(inhibition of; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Gene therapy
(modulating Egr-1 gene expression; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Antitumor agents
(sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT DNA
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(single-stranded, for targeting Egr DNA; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Neoplasm
(solid, inhibitor; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT **Antisense oligonucleotides**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(targeting Egr-1 mRNA; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT Proliferation inhibition
(vascular endothelial; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT 196222-25-0 **196222-26-1** 284057-57-4 284057-58-5
284057-59-6 284057-60-9 284057-61-0 284057-62-1 284057-64-3
337402-13-8
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT 259241-59-3
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

IT 140039-69-6 140070-40-2 140773-02-0, GenBank J04154 217884-56-5
217884-57-6 337402-33-2 337402-34-3 337402-35-4 337402-36-5
337402-37-6 337402-38-7 337402-39-8
RL: PRP (Properties)
(unclaimed nucleotide sequence; sequences of **antisense oligonucleotides** and catalytic DNA targeting Egr-1 mRNA and uses thereof in cancer therapy)

L3 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2002 ACS

IT **196222-26-1**
RL: PRP (Properties)
(unclaimed nucleotide sequence; catalytic DNA targeted to EGR-1 mRNA and their therapeutic use)

RN 196222-26-1 CAPLUS

CN DNA, d(C-T-T-G-G-C-C-G-C-T-G-C-C-A-T) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

ACCESSION NUMBER: 2000:493667 CAPLUS
DOCUMENT NUMBER: 133:116709
TITLE: Catalytic DNA targeted to EGR-1 mRNA and their therapeutic use
INVENTOR(S): Atkins, David G.; Baker, Andrew R.; Khachigian, Levon Michael
PATENT ASSIGNEE(S): Unisearch Limited, Australia; Johnson & Johnson Research Pty. Ltd.
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

ZA 9702000 A 19971024 ZA 1997-2000 19970307
EP 934404 A1 19990811 EP 1997-906032 19970307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2000506725 T2 20000606 JP 1997-531259 19970307
US 6200960 B1 20010313 US 1999-142779 19990413
PRIORITY APPLN. INFO.: AU 1996-8554 A 19960307
WO 1997-AU140 W 19970307

AB A method of inhibiting proliferation of cells by inhibiting induction or decreasing expression of the Egr-1 gene or decreasing the nuclear accumulation or activity of the Egr-1 gene product is described. Egr-1 is found to be one of the immediate-early genes induced in response to vascular injury and to play a role in restenosis and atherosclerosis. Inhibitors of Egr-1 expression may include **antisense** DNA, **ribozymes**, or transcriptional decoys. **Antisense oligonucleotides** to Egr-1 were taken by smooth muscle cells in culture without significant degrdn. and inhibited their proliferation. Egr-1 protein synthesis was inhibited, but Sp1 synthesis was not.

AB A method of inhibiting proliferation of cells by inhibiting induction or decreasing expression of the Egr-1 gene or decreasing the nuclear accumulation or activity of the Egr-1 gene product is described. Egr-1 is found to be one of the immediate-early genes induced in response to vascular injury and to play a role in restenosis and atherosclerosis. Inhibitors of Egr-1 expression may include **antisense** DNA, **ribozymes**, or transcriptional decoys. **Antisense oligonucleotides** to Egr-1 were taken by smooth muscle cells in culture without significant degrdn. and inhibited their proliferation. Egr-1 protein synthesis was inhibited, but Sp1 synthesis was not.

ST restenosis inhibition Egr1 gene; **antisense** DNA Egr1 endothelial cell proliferation; vascular smooth muscle cell proliferation Egr1

IT **Antisense** DNA
Ribozymes
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(as inhibitor of Egr-1 gene expression; control of Egr-1 synthesis and activity in inhibition of endothelial cell proliferation in control of restenosis and atherosclerosis)

IT 196222-25-0 **196222-26-1**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**antisense** DNA to Egr-1 gene for inhibition of gene expression; control of Egr-1 synthesis and activity in inhibition of endothelial cell proliferation in control of restenosis and atherosclerosis)

=> d his

(FILE 'HOME' ENTERED AT 16:52:39 ON 20 JUN 2002)

FILE 'REGISTRY' ENTERED AT 16:53:19 ON 20 JUN 2002
L1 8 S CTTGGCCGCTGCCAT/SQSN

FILE 'CAPLUS' ENTERED AT 16:55:22 ON 20 JUN 2002
L2 6 S L1
L3 4 S L2 AND ((ANTI(W)SENSE) OR ANTISENSE OR APTAMER OR TRIPLEX OR

=>

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000042173	A1	20000720	WO 2000-AU11	20000111
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151089	A1	20011107	EP 2000-902488	20000111
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: AU 1999-8103 A 19990111
WO 2000-AU11 W 20000111

AB The present invention relates to DNAzymes which are targeted against mRNA mols. encoding EGR-1 (also known as Egr-1 or NGFI-A). The present invention also relates to compns. including these DNAzymes and to methods of treatment involving administration of the DNAzymes. Thus, a DNAzyme binding to bp 189-207 of human EGR-1 mRNA and cleaving the 198G-199U bond blocked induction of EGR-1 and inhibited growth of human smooth muscle cells.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT **Ribozymes**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(deoxy; catalytic DNA targeted to EGR-1 mRNA and their therapeutic use)

IT 140070-40-2 140773-02-0, GenBank J04154 **196222-26-1**
284060-50-0 284060-51-1 284060-52-2 284060-53-3
RL: PRP (Properties)
(unclaimed nucleotide sequence; catalytic DNA targeted to EGR-1 mRNA and their therapeutic use)

L3 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2002 ACS
IT **259164-92-6**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(effect of Egr-1 inhibition by phosphorothioate **oligonucleotides** on vascular smooth muscle cell proliferation and regrowth after mech. injury in vitro)
RN 259164-92-6 CAPLUS
CN DNA, d(P-thio)(C-T-T-G-G-C-C-G-C-T-G-C-C-A-T) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
ART 1.
ACCESSION NUMBER: 1999:644873 CAPLUS
DOCUMENT NUMBER: 132:175495
TITLE: Vascular smooth muscle cell proliferation and regrowth after mechanical injury in vitro are Egr-1/NGFI-A-dependent
AUTHOR(S): Santiago, Fernando S.; Atkins, David G.; Khachigian, Levon M.
CORPORATE SOURCE: Centre for Thrombosis and Vascular Research, The University of New South Wales, Sydney, 2052, Australia

SOURCE: American Journal of Pathology (1999), 155(3), 897-905
CODEN: AJPAA4; ISSN: 0002-9440
PUBLISHER: American Society for Investigative Pathology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Smooth muscle cell (SMC) proliferation is a key event in renarrowing of blood vessels after balloon angioplasty. Mech. injury imparted to the arterial wall in exptl. models induces the expression of the immediate-early gene, egr-1. Egr-1 binds to and activates expression from the proximal promoters of multiple genes whose products can, in turn, influence the vascular response to injury. Here, we used **antisense** strategies in vitro to inhibit rat vascular SMC proliferation by directly targeting Egr-1. A series of phosphorothioate **antisense oligonucleotides** of 15 base length and complementary to various theor. accessible regions within Egr-1 mRNA were synthesized and assessed for their ability to selectively inhibit SMC proliferation in an Egr-1-dependent manner. Western blot anal. revealed that two **oligonucleotides**, AS2 and E11, inhibited Egr-1 synthesis in cells exposed to serum without affecting levels of the zinc finger protein Sp1. AS2 and E11 inhibited serum-inducible [3H]thymidine incorporation into DNA, as well as serum stimulation of total cell nos. Size-matched phosphorothioate **oligonucleotides** with random, scrambled, sense or mismatch sequences failed to inhibit. **Antisense** Egr-1 inhibition was nontoxic and reversible. These **oligonucleotides** also inhibited SMC regrowth after mech. injury in vitro. Egr-1 thus plays a key regulatory role in SMC proliferation and repair following injury.
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB Smooth muscle cell (SMC) proliferation is a key event in renarrowing of blood vessels after balloon angioplasty. Mech. injury imparted to the arterial wall in exptl. models induces the expression of the immediate-early gene, egr-1. Egr-1 binds to and activates expression from the proximal promoters of multiple genes whose products can, in turn, influence the vascular response to injury. Here, we used **antisense** strategies in vitro to inhibit rat vascular SMC proliferation by directly targeting Egr-1. A series of phosphorothioate **antisense oligonucleotides** of 15 base length and complementary to various theor. accessible regions within Egr-1 mRNA were synthesized and assessed for their ability to selectively inhibit SMC proliferation in an Egr-1-dependent manner. Western blot anal. revealed that two **oligonucleotides**, AS2 and E11, inhibited Egr-1 synthesis in cells exposed to serum without affecting levels of the zinc finger protein Sp1. AS2 and E11 inhibited serum-inducible [3H]thymidine incorporation into DNA, as well as serum stimulation of total cell nos. Size-matched phosphorothioate **oligonucleotides** with random, scrambled, sense or mismatch sequences failed to inhibit. **Antisense** Egr-1 inhibition was nontoxic and reversible. These **oligonucleotides** also inhibited SMC regrowth after mech. injury in vitro. Egr-1 thus plays a key regulatory role in SMC proliferation and repair following injury.
ST vessel smooth muscle proliferation Egr1 restenosis; angioplasty phosphorothioate **oligonucleotide** Egr1 vessel proliferation
IT Transcription factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Egr-1; effect of Egr-1 inhibition by phosphorothioate **oligonucleotides** on vascular smooth muscle cell proliferation and regrowth after mech. injury in vitro)
IT Phosphorothioate **oligonucleotides**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study);

PROC (Process)
(effect of Egr-1 inhibition by phosphorothioate **oligonucleotides** on vascular smooth muscle cell proliferation and regrowth after mech. injury in vitro)

IT Artery, disease
(restenosis; effect of Egr-1 inhibition by phosphorothioate **oligonucleotides** on vascular smooth muscle cell proliferation and regrowth after mech. injury in vitro)

IT Blood vessel
(smooth muscle; effect of Egr-1 inhibition by phosphorothioate **oligonucleotides** on vascular smooth muscle cell proliferation and regrowth after mech. injury in vitro)

IT **259164-92-6** 259164-93-7 259164-94-8 259164-95-9
259164-96-0 259164-97-1 259164-98-2 259164-99-3
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(effect of Egr-1 inhibition by phosphorothioate **oligonucleotides** on vascular smooth muscle cell proliferation and regrowth after mech. injury in vitro)

L3 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS

IT **196222-26-1**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**antisense** DNA to Egr-1 gene for inhibition of gene expression; control of Egr-1 synthesis and activity in inhibition of endothelial cell proliferation in control of restenosis and atherosclerosis)

RN 196222-26-1 CAPLUS

CN DNA, d(C-T-T-G-G-C-C-G-C-T-G-C-C-A-T) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

ACCESSION NUMBER: 1997:618194 CAPLUS
DOCUMENT NUMBER: 127:257645
TITLE: Control of Egr-1 synthesis and activity in inhibition of endothelial cell proliferation in control of restenosis and atherosclerosis
INVENTOR(S): Khachigian, Levon Michael
PATENT ASSIGNEE(S): Unisearch Ltd., Australia; Khachigian, Levon Michael
SOURCE: PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9732979	A1	19970912	WO 1997-AU140	19970307
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2248350	AA	19970912	CA 1997-2248350	19970307
AU 9720865	A1	19970922	AU 1997-20865	19970307
AU 707943	B2	19990722		

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1635txg

PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'HOME' AT 11:18:55 ON 17 JUN 2002
FILE 'HOME' ENTERED AT 11:18:55 ON 17 JUN 2002
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.84 0.84

=> file medline, biosis, caplus, embase, scisearch
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 3.15 3.15

FILE 'MEDLINE' ENTERED AT 11:25:40 ON 17 JUN 2002

FILE 'BIOSIS' ENTERED AT 11:25:40 ON 17 JUN 2002
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FILE 'CAPLUS' ENTERED AT 11:25:40 ON 17 JUN 2002
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FILE 'SCISEARCH' ENTERED AT 11:25:40 ON 17 JUN 2002
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=> egr-1
L1 5659 EGR-1

=> early growth response factor-1
L2 163 EARLY GROWTH RESPONSE FACTOR-1

=> transcription factor?
L3 281805 TRANSCRIPTION FACTOR?

=> ischemic tissue
L4 4746 ISCHEMIC TISSUE

=> repurfusion
L5 13 REPURFUSION

=> reperfusion
L6 140326 REPERFUSION

=> ls
L7 23475 LS

=> ds
L8 75909 DS

=> dh
L9 39793 DH

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=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
     17 JUN 2002
L1          5659 EGR-1
L2           163 EARLY GROWTH RESPONSE FACTOR-1
L3        281805 TRANSCRIPTION FACTOR?
L4          4746 ISCHEMIC TISSUE
L5            13 REPURFUSION
L6        140326 REPERFUSION
L7         23475 LS
L8         75909 DS
L9         39793 DH

=> del L5, L7, L8, L9
DELETE L5 L7 L8 L9? (Y)/N:y

=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
     17 JUN 2002
L1          5659 EGR-1
L2           163 EARLY GROWTH RESPONSE FACTOR-1
L3        281805 TRANSCRIPTION FACTOR?
L4          4746 ISCHEMIC TISSUE
L6        140326 REPERFUSION

=> oligonucleotide?
L9       211490 OLIGONUCLEOTIDE?

=> L1 and L2 and L3 and L4
L10             0 L1 AND L2 AND L3 AND L4

=> L1 and L2 and L4
L11             0 L1 AND L2 AND L4

=> L1 and L4 and L6
L12             5 L1 AND L4 AND L6

=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
     17 JUN 2002
L1          5659 EGR-1
L2           163 EARLY GROWTH RESPONSE FACTOR-1
L3        281805 TRANSCRIPTION FACTOR?
L4          4746 ISCHEMIC TISSUE
L6        140326 REPERFUSION
L9        211490 OLIGONUCLEOTIDE?
L10            0 L1 AND L2 AND L3 AND L4
L11            0 L1 AND L2 AND L4
L12            5 L1 AND L4 AND L6

=> del L10, L11
DELETE L10 L11? (Y)/N:y

=> d his
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divergent gene families underlying ischemic stress.
AU Yan S F; Fujita T; Lu J; Okada K; Shan Zou Y; Mackman N; Pinsky D J; Stern D M
SO NATURE MEDICINE, (2000 Dec) 6 (12) 1355-61.
Journal code: 9502015. ISSN: 1078-8956.

=> ti
L13 365396 TI

=> d ti, au, so L12 1-5

L12 ANSWER 1 OF 5 MEDLINE
TI **Egr-1**, a master switch coordinating upregulation of divergent gene families underlying ischemic stress.
AU Yan S F; Fujita T; Lu J; Okada K; Shan Zou Y; Mackman N; Pinsky D J; Stern D M
SO NATURE MEDICINE, (2000 Dec) 6 (12) 1355-61.
Journal code: 9502015. ISSN: 1078-8956.

L12 ANSWER 2 OF 5 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI **Egr-1**, a master switch coordinating upregulation of divergent gene families underlying ischemic stress.
AU Yan, Shi-Fang (1); Fujita, Tomoyuki; Lu, Jiesheng; Okada, Kenji; Zou, Yu Shan; Mackman, Nigel; Pinsky, David J.; Stern, David M.
SO Nature Medicine, (December, 2000) Vol. 6, No. 12, pp. 1355-1361. print.
ISSN: 1078-8956.

L12 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2002 ACS
TI **Egr-1**, a master switch coordinating upregulation of divergent gene families underlying ischemic stress
AU Yan, Shi-Fang; Fujita, Tomoyuki; Lu, Jiesheng; Okada, Kenji; Zou, Yu Shan; Mackman, Nigel; Pinsky, David J.; Stern, David M.
SO Nature Medicine (New York) (2000), 6(12), 1355-1361
CODEN: NAMEFI; ISSN: 1078-8956

L12 ANSWER 4 OF 5 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI **Egr-1**, a master switch coordinating upregulation of divergent gene families underlying ischemic stress.
AU Yan S.-F.; Fujita T.; Lu J.; Okada K.; Yu Shan Zou; Mackman N.; Pinsky D.J.; Stern D.M.
SO Nature Medicine, (2000) 6/12 (1355-1361).
Refs: 50
ISSN: 1078-8956 CODEN: NAMEFI

L12 ANSWER 5 OF 5 SCISEARCH COPYRIGHT 2002 ISI (R)
TI **Egr-1**, a master switch coordinating upregulation of divergent gene families underlying ischemic stress
AU Yan S F (Reprint); Fujita T; Lu J S; Okada K; Zou Y S; Mackman N; Pinsky D J; Stern D M
SO NATURE MEDICINE, (DEC 2000) Vol. 6, No. 12, pp. 1355-1361.
Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707.
ISSN: 1078-8956.

=> d his

(FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON 17 JUN 2002
L1 5659 EGR-1
L2 163 EARLY GROWTH RESPONSE FACTOR-1

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     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
     17 JUN 2002
L1          5659 EGR-1
L2           163 EARLY GROWTH RESPONSE FACTOR-1
L3        281805 TRANSCRIPTION FACTOR?
L4          4746 ISCHEMIC TISSUE
L6        140326 REPERFUSION
L9        211490 OLIGONUCLEOTIDE?
L12            5 L1 AND L4 AND L6

=> d ti, au, so

L12  ANSWER 1 OF 5     MEDLINE
TI   Egr-1, a master switch coordinating upregulation of
     divergent gene families underlying ischemic stress.
AU   Yan S F; Fujita T; Lu J; Okada K; Shan Zou Y; Mackman N; Pinsky D J; Stern
     D M
SO   NATURE MEDICINE, (2000 Dec) 6 (12) 1355-61.
     Journal code: 9502015. ISSN: 1078-8956.


=> d ti, au, so/all
'SO' MUST END IN '/Q', '/A', '/L', '/S' OR '/B'
The saved name for a query (or structure or screen set) must end with
'/Q'.  The saved name for an answer set must end with '/A'.  The saved
name for an L# list must end with '/L'.  SDI request names must end
with '/S'.  To see a list of all saved query, answer set,, and L# list
names for this loginid, enter "DISPLAY SAVED" at an arrow
prompt (=>).  Enter "DISPLAY SAVED/S" to see a list of SDI request
names.  Enter "DISPLAY SAVED/B" to see a list of BATCH search
requests.

=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
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L3        281805 TRANSCRIPTION FACTOR?
L4          4746 ISCHEMIC TISSUE
L6        140326 REPERFUSION
L9        211490 OLIGONUCLEOTIDE?
L12            5 L1 AND L4 AND L6

=> d ti, au, so L12/1-5
'L72' MUST END IN '/Q', '/A', '/L', '/S' OR '/B'
The saved name for a query (or structure or screen set) must end with
'/Q'.  The saved name for an answer set must end with '/A'.  The saved
name for an L# list must end with '/L'.  SDI request names must end
with '/S'.  To see a list of all saved query, answer set,, and L# list
names for this loginid, enter "DISPLAY SAVED" at an arrow
prompt (=>).  Enter "DISPLAY SAVED/S" to see a list of SDI request
names.  Enter "DISPLAY SAVED/B" to see a list of BATCH search
requests.

=> d ti, au, so L12

L12  ANSWER 1 OF 5     MEDLINE
TI   Egr-1, a master switch coordinating upregulation of
```

```
L3       281805 TRANSCRIPTION FACTOR?
L4         4746 ISCHEMIC TISSUE
L6       140326 REPERFUSION
L9       211490 OLIGONUCLEOTIDE?
L12           5 L1 AND L4 AND L6
L13      365396 TI

=> del L13
DELETE L13? (Y)/N:y

=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
     17 JUN 2002
L1          5659 EGR-1
L2           163 EARLY GROWTH RESPONSE FACTOR-1
L3       281805 TRANSCRIPTION FACTOR?
L4         4746 ISCHEMIC TISSUE
L6       140326 REPERFUSION
L9       211490 OLIGONUCLEOTIDE?
L12           5 L1 AND L4 AND L6

=> rd L12
MISSING OPERATOR RD L12
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> dup rem
ENTER L# LIST OR (END):L12
PROCESSING COMPLETED FOR L12
L13           1 DUP REM L12 (4 DUPLICATES REMOVED)
                ANSWER '1' FROM FILE MEDLINE

=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
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L6       140326 REPERFUSION
L9       211490 OLIGONUCLEOTIDE?
L12           5 L1 AND L4 AND L6
L13           1 DUP REM L12 (4 DUPLICATES REMOVED)

=> L1 and L9 and L6
L14           1 L1 AND L9 AND L6

=> d ti, au, so

L14  ANSWER 1 OF 1     MEDLINE
TI   Gene expression profile in mouse myocardium after ischemia.
AU   Lyn D; Liu X; Bennett N A; Emmett N L
SO   PHYSIOLOGICAL GENOMICS, (2000 Apr 27) 2 (3) 93-100.
     Journal code: 100894125. ISSN: 1094-8341.


=> d his
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     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
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L3        281805 TRANSCRIPTION FACTOR?
L4          4746 ISCHEMIC TISSUE
L6        140326 REPERFUSION
L9        211490 OLIGONUCLEOTIDE?
L12            5 L1 AND L4 AND L6
L13            1 DUP REM L12 (4 DUPLICATES REMOVED)
L14            1 L1 AND L9 AND L6

=> L3 and L9
L15       11683 L3 AND L9

=> L15 and L4 and L6
L16            3 L15 AND L4 AND L6

=> d ti, au, so 1-3

L16  ANSWER 1 OF 3     MEDLINE
TI   Oxidant stress activates AP-1 and heparin-binding epidermal growth
     factor-like growth factor transcription in renal epithelial cells.
AU   Sakai M; Tsukada T; Harris R C
SO   EXPERIMENTAL NEPHROLOGY, (2001) 9 (1) 28-39.
     Journal code: 9302239. ISSN: 1018-7782.

L16  ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2002 ACS
TI   Oxidant stress activates AP-1 and heparin-binding epidermal growth
     factor-like growth factor transcription in renal epithelial cells
AU   Sakai, Masahiro; Tsukada, Toshiaki; Harris, Raymond C.
SO   Experimental Nephrology (2000), Volume Date 2001, 9(1), 28-39
     CODEN: EXNEEG; ISSN: 1018-7782

L16  ANSWER 3 OF 3  EMBASE  COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI   Oxidant stress activates AP-1 and heparin-binding epidermal growth
     factor-like growth factor transcription in renal epithelial cells.
AU   Sakai M.; Tsukada T.; Harris R.C.
SO   Experimental Nephrology, (2001) 9/1 (28-39).
     Refs: 69
     ISSN: 1018-7782  CODEN: EXNEEG


=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
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L12            5 L1 AND L4 AND L6
L13            1 DUP REM L12 (4 DUPLICATES REMOVED)
L14            1 L1 AND L9 AND L6
L15        11683 L3 AND L9
L16            3 L15 AND L4 AND L6

=> print
```

```
ENTER (L16), L#, OR ACC:acc
'ACC' NOT VALID WITH MULTIFILE PROCESSING

=> d his

     (FILE 'HOME' ENTERED AT 11:16:11 ON 17 JUN 2002)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, SCISEARCH' ENTERED AT 11:25:40 ON
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L12            5 L1 AND L4 AND L6
L13            1 DUP REM L12 (4 DUPLICATES REMOVED)
L14            1 L1 AND L9 AND L6
L15        11683 L3 AND L9
L16            3 L15 AND L4 AND L6
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